Modern Concepts of Cardiovascular Disease

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SYNCOPE DUE TO CARDIAC ARRHYTHMIAS*

Syncope can be defined as a sudden transient suspension of consciousness. The type being discussed here is due to a *sudden* decrease in cerebral blood flow resulting from an inadequate cardiac output secondary to the various cardiac arrhythmias. Syncope that is not primarily due to an abnormal cardiac rhythm is not discussed, and for this reason no comments are included regarding the common faint, postural hypotension, etc. The variables which determine the severity of the attacks are: 1. The abruptness, degree, and duration of inadequate cardiac output. 2. The status of the cerebral arteries. Accordingly, the symptoms may vary from momentary giddiness to loss of consciousness sufficient to cause injury. Severe and prolonged attacks associated with convulsions are more common in elderly patients because of associated cerebral atherosclerosis.

ARRHYTHMIAS WHICH CAN PRODUCE SYNCOPE

Paroxysmal auricular tachycardia or *fibrillation* occasionally produce syncope in children and young adults. Under such circumstances the transient loss of consciousness occurs at the onset of the attack before the body can adapt to the rapid heart rate and at the end of the attack when there is a brief period of ventricular arrhythmia or cardiac standstill. Syncope as a result of supraventricular tachycardia is even more likely to occur in older folks with cerebral atherosclerosis, and here too it tends to occur at the onset and at the end of an attack of rapid beat. Normal sinus rhythm may be interrupted by *ventricular tachycardia* or by *ventricular fibrillation*, and this change in rhythm may produce syncope. One of the causes of syncope in patients with *carotid sinus syndrome* is a disturbance in cardiac rhythm which may be total standstill of the atria and ventricles or high grade atrioventricular block. Syncope may be associated with varying degrees of *atrioventricular block*. Morgagni reported the case of "a grave and worthy priest" who was "seized with the epilepsy which left behind it the greatest *slowness* of the pulse." Adams reported the case of "an officer in the revenue" who had a "remarkable *slowness* of the pulse which generally ranged at the rate of 30 in a minute." Stokes studied several cases and reported his "Observations on Some Cases of Permanently Slow Pulse." Since all three of these physicians observed cases of unusually slow pulse, it seems desirable to limit the use of the term Morgagni-Stokes-Adams syndrome to cases of syncope associated with atrioventricular block.

The following mechanisms may be responsible for the attack in such patients: 1. Complete atrioventricular block and ventricular asystole may interrupt normal sinus rhythm (with or without prior evidence of A-V conduction disturbance), or ventricular asystole may occur suddenly in a case of complete heart block. These mechanisms are responsible for syncope in approximately 55 per cent of the cases associated with atrioventricular block. 2. Patients with complete heart block may develop ventricular tachycardia or fibrillation which may or may not be followed by ventricular standstill. These mechanisms are responsible for 45 per cent of the attacks. 3. When there is complete heart block, the idioventricular focus may, on rare occasions, gradually slow below a critical level and produce syncope. 4. One of the above mechanisms may be responsible for an attack on one occasion, whereas some other mechanism may be responsible on another occasion.

The tendency of some to call all cases of cardiac syncope "Morgagni-Stokes-Adams" attacks has led to confusion. Since the treatment and prognosis varies with the mechanism responsible for the episode, it is necessary to describe the mechanism when it is known.

ETIOLOGY

Paroxysmal auricular tachycardia is common in subjects without evidence of heart disease but also occurs with mitral stenosis, thyrotoxicosis, and myocarditis. *Paroxysmal auricular fibrillation* and flutter occur in patients without evidence of heart disease but also occur in mitral stenosis, thyrotoxicosis, pulmonary embolism, constrictive pericarditis, and coronary disease, and may be precipitated by pulmonary infections and surgical procedures. *Ventricular tachycardia* and *fibrillation* occur with acute coronary occlusion, chronic coronary disease, aortic stenosis, aortic insuffi-

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ciency, and myocarditis. These conditions can also be produced by digitalis, quinidine, and by procaine amide (Pronestyl®) intoxication.

Morgagni-Stokes-Adams attacks usually occur in patients who have disease of the atrioventricular conduction system (A-V node and bundle) secondary to coronary atherosclerosis. This abnormality in conduction may be present even though there is no history of angina pectoris or evidence of myocardial infarction since the larger coronary vessels may not be diseased sufficiently to cause these more common manifestations of coronary atherosclerosis. It is not unusual to observe an increase in P-R interval, Wenckebach phenomenon, and complete heart block, with or without syncope, as a complication of acute posterior myocardial infarction. These disturbances occur because the usual posterior infarct is caused by thrombosis of the right coronary artery or circumflex branch of the left coronary artery, and branches from these vessels also supply the A-V conduction system. Under these circumstances the A-V block is usually transient. Atrioventricular block and Morgagni-Stokes-Adams attacks may occur in patients with calcific aortic stenosis. It is interesting to note that the case reported by Stokes and several of the cases reported by Adams had aortic stenosis. There are several causes for heart block in such cases. The calcific process may extend into the conduction pathway in some cases while coronary insufficiency may be responsible for the damage in others. Acute rheumatic fever and diphtheria may produce high grade A-V block, but actual syncope is rare. Now that electrocardiograms are readily available, it has become apparent that many infectious diseases, such as mumps, measles, and scarlet fever, are associated with electrocardiographic abnormalities including various degrees of A-V block, but syncope is very rare. The A-V conduction pathway may be damaged by gummata, tuberculosis, neoplasms, trauma, and by bacterial endocarditis. Complete A-V block may be congenital in origin and is occasionally associated with an interventricular septal defect, but syncope seldom occurs. All grades of A-V block can be produced by digitalis. In addition, the drug may augment forces already present that are tending to produce A-V block. Quinidine can also produce A-V conduction disturbance. Reflex vagal stimulation from pressure on a sensitive carotid sinus, swallowing, coughing, and straining can produce or contribute to A-V block.

CLINICAL MANIFESTATIONS

1. Observations between attacks of syncope. Patients may have complaints secondary to their basic heart disease but may or may not complain of symptoms relative to the heart beat between the episodes of syncope. Some will be aware of ectopic cardiac beats, and the patient with persistent complete heart block may be conscious of the unusually slow, but strong, cardiac contractions. The physical examination is frequently normal, but, if mitral stenosis is found, one should suspect paroxysmal auricular fibrillation. If aortic stenosis is found, one should suspect several mechanisms. Thus, Schwartz[1] has observed that the most common cause of syncope in aortic stenosis is a gradual slowing of the ventricular rate; however, ventricular fibrillation and complete A-V block with ventricular asystole has also been observed.

Since syncope may occur in patients with atrioventricular conduction disturbance, it is important to keep in mind the physical findings associated with all grades of block. This is especially important when observing the patient with posterior myocardial infarction. The intensity of the first heart sound depends, to some degree at least, on the previous P-R interval. Whenever the P-R interval is prolonged, the apical first sound tends to be decreased in intensity. When the Wenckebach phenomenon is present, the apical first sound varies from cycle to cycle and the long diastolic pauses are not preceded by premature contractions. When the cardiac rate is as slow as 40 to 60 beats per minute, one should consider sinus bradycardia, 2:1 heart block, and complete heart block. Sir Thomas Lewis[2] pointed out that "in complete heart block the action of the ventricles is phenomenally slow, nearly all hearts which beat at rates of 35 and under are affected in this manner. . . . Each ventricular beat is accompanied by a first and second sound; in addition, very faint muffled sounds are usually to be heard in the long diastoles. . . . The latter are due to auricular systoles. . . . A sign which is conclusive, and almost always present, is a changed character of the first and second heart sounds from beat to beat. . . . From time to time a prominent venous pulsation accompanies the intensified first heart sound, when auricular systole coincides with that of the ventricle." Levine and Harvey[3] have stressed these findings also and state that a changing intensity of the first heart sound is almost pathognomonic of complete heart block when the heart rate is unusually slow. Occasionally one is able to feel a weak pulsation at the wrist due to an auricular pulsation which has been transmitted directly to the peripheral arteries. When auricular fibrillation is present, certain of the previous signs will vanish since they depend on normal auricular contractions.

Between episodes of cardiac syncope the electrocardiogram may or may not be helpful. Any of the previously listed cardiac arrhythmias may occur paroxysmally and the electrocardiogram can be normal between episodes. Helpful clues are as follows: When there is Wolff-Parkinson-White syndrome or frequent auricular premature contractions in the electrocardiogram between attacks, the chances of the episodes being paroxysmal auricular tachycardia or fibrillation are great. Patients who have a short P-R interval associated with normal QRS duration are also

subject to episodes of paroxysmal auricular tachycardia. Where there are multifocal ectopic ventricular contractions noted, ventricular tachycardia or fibrillation may be the cause of the episodes. This is particularly true when the ventricular ectopic contraction arises "out of the T waves" or occur in bursts of three or four in succession. If there is evidence of atrioventricular conduction disturbance, then one of the arrhythmias complicating heart block is more likely to be responsible for the episodes.

2. Observations during attacks of syncope. The mothers of infants with paroxysmal auricular tachycardia can be taught to observe the chest and upper abdomen for rapid pulsations during episodes of loss of consciousness. At times the adult patient can actually observe a few moments of sudden paroxysmal rapid heart action prior to and after momentarily "blacking out." Occasionally the physician can make certain observations during an attack. If, by chance, he is present at the time of the attack of syncope due to paroxysmal rapid heart beat, he may be able to establish the cardiac mechanism by observations made just before or after the attack.

Paroxysmal auricular tachycardia produces a regular rate of 160 to 250, and the first heart sound does not vary from cycle to cycle. The rapid beat is either unaffected by carotid sinus pressure or the abnormal rate returns abruptly to and remains normal. One should be suspicious of auricular flutter with 2:1 block when the heart rate suddenly reaches the vicinity of 150. Carotid sinus pressure increases the block in cases of auricular flutter and slows the rate, but, when the pressure ceases, the apex beat "jerks" back to the previous rate. Auricular fibrillation is characterized by an irregular irregularity and a pulse deficit. Ventricular tachycardia begins suddenly, and there may be slight variation in the intensity of the first heart sound. The rate may be 90 to 200; it is unaffected by carotid sinus pressure. When syncope is due to ventricular fibrillation or standstill, no heart sounds are heard and accurate differentiation cannot be made by physical examination.

Since one is seldom fortunate enough to record an electrocardiogram during the actual episode of syncope, one is forced to base his opinion regarding the responsible underlying cardiac mechanism on circumstantial evidence. It is necessary to consider the age of the patient; the type of underlying heart disease (if present); the symptoms of the patient prior to or after the transient loss of consciousness; and the electrocardiogram between attacks.

TREATMENT

An attack of syncope secondary to an abnormal heart rhythm can seldom be treated because of its short duration. Attacks can sometimes be prevented, however, by therapy designed to prevent the episodes of cardiac arrhythmia.

The treatment of an attack of paroxysmal auricular tachycardia is well established. If carotid sinus pressure does not abort an attack, the drug of choice is digitalis unless the arrhythmia is actually due to digitalis. Lanatocide C (1.6 milligrams) or digoxin (1.5 milligrams of the new preparation in propylene glycol) should be given intramuscularly to adults not on digitalis. Some physicians give one-half the listed dose of these drugs cautiously intravenously and repeat if necessary in one to two hours. Carotid sinus pressure should be tried again thirty to sixty minutes after the digitalis has been given. If digitalis fails to revert the attack to normal rhythm, then quinidine sulfate can be used. A maintenance dose of digitalis may be successful in preventing attacks, but, if it fails, then quinidine sulfate should be employed.

Paroxysmal auricular fibrillation or flutter with an uncontrolled ventricular rate is also treated with digitalis. It is not commonly understood that the object of treatment initially is to slow the ventricular rate. Digitalis is very reliable in accomplishing this. In addition some cases of paroxysmal auricular fibrillation will be reverted to normal rhythm by digitalis medication. This is more likely to occur when auricular fibrillation is not associated with mitral stenosis or thyrotoxicosis. The urgency of the situation determines whether one uses a rapid acting intravenous preparation or an oral preparation of digitalis. After the ventricular rate has been brought under control, quinidine sulfate may be used to revert the rhythm to normal if such is desired. Quinidine sulfate and/or digitalis can be used to prevent the paroxysms of auricular fibrillation and flutter. Obviously thyrotoxicosis must be treated when it is the cause of the auricular fibrillation.

Ventricular tachycardia is treated with either procaine amide or quinidine. Procaine amide can be given intramuscularly in a dose of 0.5 to 1.0 gram every two hours for several doses. One gram of procaine amide may be diluted in 100-200 cc. of glucose and given intravenously over a period of one half to one hour. Norepinephrine (Levophed®) can be given to counteract the hypotension that is frequently associated with the use of procaine amide intravenously. If procaine amide fails, quinidine gluconate (0.4-0.6 gram) can be given intramuscularly every two hours for several doses. Quinidine gluconate should not be used intravenously except in desperate situations. All therapy must be monitored by frequent electrocardiograms. Quinidine sulfate and procaine amide are useful in preventing recurrent episodes of ventricular tachycardia.

One seldom has the opportunity to treat an episode of ventricular fibrillation. As a rule the episode ends spontaneously, or, if it is continuous, it produces death. When the opportunity does arise, the treatment with procaine amide intravenously is usually to no avail. When there are short repeated bouts of ventricular fibrillation

which interrupt normal sinus rhythm, procaine amide or quinidine may be used in an attempt to prevent the episodes.

The therapy for the cardiac mechanism responsible for syncope in patients with atrioventricular block is unsatisfactory; however, several recent important observations have been made. Therapy is aimed at *preventing episodes of syncope.* For example, one does not treat a patient with established complete heart block when he is asymptomatic. Indeed, it is questionable whether or not therapy is justified in such a patient when the episodes of syncope are infrequent or have occurred in the distant past. Obviously the first step in therapy is to eliminate all sources which will tend to produce vagatonia. Digitalis may contribute to the A-V block and its use, even with great caution, is justified only if there is congestive heart failure. When the electrocardiogram between episodes of syncope shows high grade A-V conduction disturbance, it is useful to establish the benefit, or lack of benefit, of atropine sulfate by giving one milligram intravenously. Though the drug is usually of no benefit, if the electrocardiogram shows a lessening of the A-V block after the drug has been given, atropine should be given by mouth several times a day.

Ephedrine has been used to prevent episodes of syncope due to ventricular standstill in cases with evidence of A-V block. When episodes are very frequent, then intramuscular injections of adrenalin (0.25 to 1.0 cc. of 1:1000) are given at frequent intervals. If one is present at the time of prolonged ventricular standstill, adrenalin can be given directly into the heart. Isopropylarterenol (Isuprel®)[4] can be used to prevent the ventricular asystole associated with A-V block. This drug apparently blocks the carotid sinus reflex and complete A-V block may be changed to lesser grades of block. Isopropylarterenol also increases the rate of the idioventricular pacemaker in complete heart block and tends to prevent ventricular asystole secondary to spontaneous slowing of the pacemaker. Isopropylarterenol is better tolerated in older folks than adrenalin since it causes much less cerebral stimulation and less pressure response. There is also some evidence indicating that isopropylarterenol is less likely to predispose to ventricular fibrillation than is adrenalin. Five to ten milligrams should be administered sublingually every two to six hours depending on the frequency of attacks. An electrical cardiac pacemaker[5] may be employed when the periods of ventricular asystole are extremely frequent. In this technique an electric current is applied to the closed chest by means of two electrodes. The amount of current needed to produce a ventricular response is determined in each individual case, and then the stimulus is applied at an effective rate. It is hoped, in such circumstances, that the idioventricular pacemaker will become established and that the electric pacemaker can be discontinued. Quinidine sulfate and procaine amide are contraindicated in cases of asystole associated with A-V block.

The treatment for transient ventricular fibrillation during established heart block is still unknown. It has become apparent that quinidine[6] and procaine amide frequently *aggravate* the condition. The bouts of ventricular fibrillation may become more frequent and the period of standstill that occasionally follows the ventricular fibrillation may become more prolonged after these drugs. Adrenalin also aggravates the condition by precipitating more frequent episodes of ventricular fibrillation. At present some investigators[1] believe that isopropylarterenol is also contraindicated. Others[7] believe that isopropylarterenol decreases the number of episodes of paroxysmal ventricular fibrillation and tachycardia in cases with established heart block. To date, the treatment is unsatisfactory but isopropylarterenol should be tried with great caution.

SUMMARY

There are numerous cardiac arrhythmias that may, under certain circumstances, produce syncope. The etiology and clinical recognition of these arrhythmias have been discussed. Since the definition of Morgagni-Stokes-Adams attack is not universally the same, a plea is made to describe the abnormal cardiac mechanism which causes the syncope. The treatment has been discussed in some detail and may be simple at times but is often unsatisfactory. Attention is called to the fact that a drug may be indicated for an arrhythmia on one occasion whereas, under different circumstances, the drug may be contraindicated for the same arrhythmia. For example, quinidine and procaine amide may prevent episodes of ventricular fibrillation from occurring in patients with normal sinus rhythm but these same drugs are contraindicated in patients who have transient ventricular fibrillation associated with heart block. Some advantages of isopropylarterenol in preventing episodes of ventricular standstill associated with A-V block have been pointed out.

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The opinions and conclusions expressed herein are those of the author and do not necessarily represent the official views of the Scientific Council of the American Heart Association.

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